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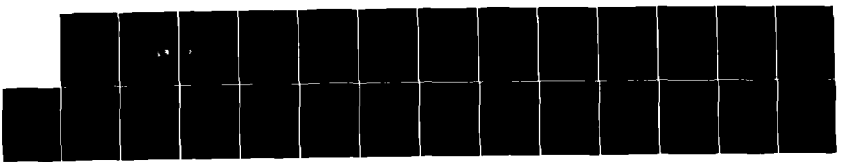

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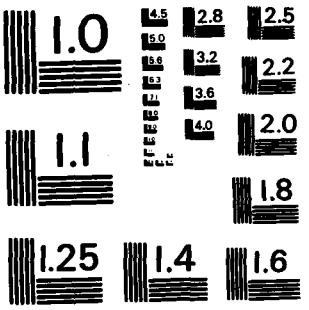

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Peak torque decreased 67 (range 47-82) % as a result of the repeated contractions. Following recovery peak torque averaged 70 (47-86)% of the initial value.

Lactate concentration after the 50 contractions was 2.9 $\pm$ 1.3 mmol.l^{-1} and the peak post exercise value averaged 8.7 $\pm$2.1 mmol·l mmol·l^{-1} Fatigue and recovery respectively were correlated with capillary density (r=0.71 and 0.69) but not with fiber type distribution. A relationship was demonstrated between capillary density and the calculated lactate release (r-o.64). Based on the present findings it is suggested that lactate elimination from the exercising muscle is dependent upon the capillary supply and influences the rate of muscle force recovery.

To Dr. Jan Vogel best regards / PER
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RECOVERY FROM SHORT TERM INTENSE EXERCISE: ITS RELATION TO CAPILLARY SUPPLY AND LACTATE RELEASE

By

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Short title: Fatigue, recovery and lactate

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Summary. Muscle force recovery from short term intense exercise was examined in 16 physically active men. They performed 50 consecutive maximal voluntary knee extensions. Following a 40 s rest period 5 additional maximal contractions were executed. The decrease in torque during the 50 contractions and the peak torque during the 5 contractions relative to initial torque were used as indicies for fatigue and recovery, respectively. Venous blood samples were collected repeatedly up to 8 min past exercise for subsequent lactate analyses. Muscle biopsies were obtained from m. vastus lateralis and analysed for fiber type composition, fiber area and capillary density.

Peak torque decreased 67 (range 47-82)% as a result of the repeated contractions. Following recovery peak torque averaged 70 (47-86)% of the initial value.

Lactate concentration after the 50 contractions was 2.9±1.3 mmol·l^{-1} and the peak post exercise value averaged 8.7±2.1 mmol·l^{-1}. Fatigue and recovery respectively were correlated with capillary density (r=0.71 and 0.69) but not with fiber type distribution. A relationship was demonstrated between capillary density and the calculated lactate release (r=0.64). Based on the present findings it is suggested that lactate elimination from the exercising muscle is dependent upon the capillary supply and influences the rate of muscle force recovery.

Key words: fatigue; mean fiber area; muscle fiber types; capillary density; peak torque

Introduction

Heavy muscular exercise may result in impairment of the potential for further exercise. The reduction in performance capacity, caused by previous exercise, depends upon the mode, intensity and duration of exercise as well as the length of recovery phase. Accumulation of lactate and/or associated shifts in pH have been regarded as the most likely factors responsible for this impairment (cf. Simonson 1971; Karlsson 1979; Tesch 1980). It has been speculated that lactate and an associated increase in H^+ interfere with the formation of cross-bridges in the muscle cell by competing with the Ca^{++} on the binding sites of troponin (Nakamura and Schwartz 1972; Robertson and Kerrick 1979). Both animal studies (Beatty et al. 1963; Baldwin and Tipton 1972) and recent findings in humans (Tesch et al. 1978; Tesch 1980) have suggested lactate to accumulate at a higher rate in fast twitch (FT or Type II) than in slow twitch (ST or Type I) fibers of exercised muscles. Lactate formed and accumulated in FT fibers may accordingly result in a greater inhibitory effect on the contractile machinery and a greater decrement in muscle force if the muscle is made up by a high percentage of FT fibers than can be expected in a "slow twitch" muscle. Moreover, the over-all metabolic profile of the ST fiber speaks in favor of a higher potential to recover from previous exercise, since one can assume that a ST muscle in comparison to a FT muscle is more efficient in oxidizing lactate due to differences in LDH enzyme and isozyme patterns (Sjödin 1976). Moreover, a higher rate of

lactate release to the blood stream may be expected from ST fibers due to a more developed capillary network (Folkow and Halicka 1968; Andersen and Henriksson 1977).

The following study was conducted to examine the recovery and lactate elimination pattern with regard to the capillary supply of exercising muscle following a short term, intense local performance task (Thorstensson and Karlsson 1976) which has been shown to induce reduction in maximal voluntary strength.

Subjects, methods and materials

Sixteen male subjects volunteered for the study. All were informed of the purpose and the risks associated with the experiments, prior to giving their written consent. The subject group represented a variety in terms of physical activity background including endurance running and advanced strength training programs as well as more general training. Age, height, weight, and maximal oxygen uptake attained on cycle ergometer averaged (±SD) 27±4 yrs, 176±5 cm, 78.5±9.8 kg and 49±8 $ml \cdot kg^{-1} \cdot min^{-1}$.

The first visit to the laboratory was intended to familiarize subjects with the isokinetic device (Cybex II® Lumex Inc., N.Y.) and the protocol to be used in the experiments. Peak torque, produced at angular velocities corresponding to 30^{o}, 180^{o} and $300^{o} \cdot s^{-1}$, was recorded during knee extension. After a short rest period a fatigue test consisting of 50 consecutive, maximal voluntary contractions performed at $180^{o} \cdot s^{-1}$, was conducted. For further information concerning the experimental set-up, see Thorstensson and Karlsson (1976) and Tesch (1980).

Two days later the actual experiments were carried out. The fatigue test was repeated and following a 40 s rest period five additional consecutive, maximal voluntary contractions were executed. The relative decrease in torque during 50 contractions was used as an index for fatigue. Day to day variation for the fatigue test amounted to 2.6% (coefficient of variation). Peak torque, developed during the following five contractions, relative to initial torque was calculated and expressed as an index for recovery (Fig. 1).

Blood samples for spectrophotometric determination of lactate concentration (Sigma Techn. Bull. 826, 1968) were obtained through an indwelling catheter from an antecubital vein prior to exercise, during the short recovery period, immediately after the additional five contractions, and at each minute up to eight minutes post exercise. As an index of lactate release the concentration immediately post exercise relative to peak lactate concentration was calculated (Fig. 1).

Muscle biopsies (Bergström 1962) were obtained from m. vastus lateralis of the investigated limb at rest for subsequent histochemical analysis. Tissue specimen were frozen in liquid nitrogen-cooled isopentane and stainings were performed on cross-sections for myofibrillar ATPase (Padykula and Herman 1955), NADH-tetrazolium reductase (Novikoff et al. 1961) and Amylase-PAS (Andersen and Henriksson 1977). Muscle fiber type distribution (%FT, %FT area) and mean muscle fiber area were calculated according to Tesch (1980) while capillary density expressed as $cap \cdot mm^{-2}$ and $cap \cdot fib^{-1}$ were assessed as described elsewhere (Andersen and Henriksson 1977).

Results

Values for peak torque per kg body weight at 30, 180 and $300^{o} \cdot s^{-1}$, initial peak torque, peak torque decrease during the fatigue test, increase in peak torque during recovery are presented in Table 1 along with information on fiber type distribution, fiber area and capillary density. The inter-relationships between peak torque at different angular velocities and mean fiber area and fiber type distribution are depicted in Table 2.

Mean initial peak torque at $180^{o} \cdot s^{-1}$ was 178 (102-245) Nm. Peak torque decreased on average of 120 (range 61-201) Nm or 67 (47-82) % following the 50 consecutive contractions. Peak torque following recovery was 70 (47-86) % of the initial value.

Blood lactate concentration at rest, following the 50 contractions and immediately following the final contractions averaged (±SD) 1.5±0.3, 2.9±1.3 and 5.3±2.3 $mmol \cdot l^{-1}$. The peak value following exercise was 8.7±2.1 $mmol \cdot l^{-1}$ (Fig. 1). Lactate release immediately following the 5th contraction relative to peak lactate concentration after exercise was 62±21% (Fig. 2).

Peak torque decrease (fatigue) was negatively correlated with recovery ($r=-0.64$, $p<0.01$). Neither fatigue nor recovery were correlated with fiber type distribution. Fatigue and recovery respectively were, however, correlated with $cap \cdot mm^{-2}$ ($r=0.71$, $p<0.01$ and $r=0.69$, $p<0.01$, Fig. 3). Positive relationships were also present between $cap \cdot mm^{-2}$ and lactate release

($r=0.64$, $p<0.01$, Fig. 4) and lactate release and recovery ($r=0.55$, $p<0.05$).

Multiple regression analysis revealed that 95% ($R=0.98$) of the variance in recovery from fatigue could be explained by fiber type distribution, capillary density, fatigue and peak torque.

Discussion

Exercise induced muscle impairment, measured as a decline in voluntary muscle strength, seems to have several causes and sites of origin (cf. Asmussen 1979; Edwards 1981; Karlsson 1979; Tesch 1980). Even though the occurrence of "central fatigue" or inhibition of parts of the central nervous system (CNS) may result in deteriorated muscle function, the results from a majority of studies suggest that metabolic events taking place within the muscle fiber are the principal contributing factors to the development of muscle fatigue during short term high intensity exercise. As a third alternative or possibly working in concert with pure "muscle fatigue" and "CNS fatigue", a feed-back mechanism involving the muscle and higher centers of the nervous system has been proposed (Asmussen and Mazin 1978a, b). Nevertheless, a hypothetical model to explain muscle function impairment, following intense exercise under conditions of augmented glycogenolytic activity, can be sketched. With extraordinarily high energy requirement and in the absence of molecular oxygen lactic acid is produced and accumulated in the muscle at a high rate concomitant with a decreased muscle pH. The presence of an increased number of hydrogen ions within the muscle cell causes an inhibition of cross-bridge formation, since hydrogen competes with and seems to have higher affinity to troponin than calcium (cf. Hermansen 1981). Hence, with less binding sites available for calcium fewer interactions between actin and myosin can be established and the muscle tension produced is subsequently reduced (Nakamura and Schwartz 1972; Robertson and Kerrick 1979).

In contrast to previous studies (Thorstensson and Karlsson 1976; Tesch 1980) using the same experimental set-up no significant relation was present between force decline and the relative distribution of FT fibers. Hence, it is obvious that the fiber type - fatigue relationship can be altered.

Animal studies (Kugelberg and Edström 1968; Kugelberg and Lindegren 1979) have demonstrated that fatigue properties of single motor units were related to the oxidative capacity of the innervated muscle fibers. Recently, Ivy et al. (1982), using a protocol similar to our own, were able to demonstrate a relationship (r=0.63) between the respiratory capacity of the exercising muscle and rate of fatigue development whereas lower correlations were established between fiber type composition and muscle fatigue. The finding here of a relationship between fatigue index and capillary density could be interpreted in the same way. That is, the capillary supply reflects the over-all metabolic profile of the exercising muscle and parallels both mitochondrial content (Brodal et al. 1976) and the activity of enzymes regulating oxidation (Hudlická, 1981).

In the present study voluntary muscle strength was found to decrease by an average of 67% and after a brief recovery period contractile function was still depressed by 30%. To a certain extent recovery in muscle contractility was influenced by the magnitude of the preceding reduction in muscle strength. Individuals who were most susceptible to fatigue also exhibited the smallest recovery in muscle force.

Using an identical fatigue protocol, pronounced muscle lactate accumulation occurred and concentrations remained the same

or decreased only slightly 30 s post exercise (Tesch 1980). In that study, however, a rapid restitution of muscle force took place. Also, blood lactate concentration after exercise was lower in subjects with muscles which decreased most in muscle force, rich in FT fibers and producing most lactate. It was speculated that individual variations in capillary supply of the exercising muscle had impact on elimination of lactate from the muscle. The fact that recovery from short term intense exercise was shown to be correlated with the density of the capillary bed is thus supportive.

There are at least two possible explanations for the relationship between capillary supply on one hand and recovery and lactate release on the other. First, capillary supply is simply paralleling the oxidative capacity of the muscle. As an alternative proposition capillary density is a true reflection of the open capillary surface area and thus the blood transport system. Support for the first assumption was found in that the magnitude of recovery, using the present experimental procedures, was correlated with citrate synthase activity, a marker for oxidative capacity (Tesch et al. 1983). Thus the possible role of oxidative metabolism in recovery processes (Nassar-Gentina et al. 1978) was implied and since fatigue index was not negatively related to the activity of this enzyme a causal relationship does not seem implausible. Jorfeldt and co-workers (1978) found the rate of lactate release from exercising muscle to blood to be maximal at muscle lactate concentrations of approximately 5 $mmol \cdot kg^{-1}$w.w in spite of a further increased blood flow. Thus membrane transport or uptake and elimination

by the vascular system seemed to be impeded above that lactate level. The authors did not indicate the magnitude of the individual range in this response. It is therefore impossible to speculate whether the levelling off observed was influenced by peripheral factors such as capillary supply.

From the present findings we cannot elucidate which of these two factors exerted the greatest influence on recovery factors. However, it can be concluded that muscle force recovers at a faster rate than lactate is eliminated.

Dynamic muscle strength strongly correlated with the relative area occupied by FT fibers rather than with muscle fiber size irrespective of contraction speed (angular velocity). Mean fiber area of a muscle has been proposed to be a reliable measure of its total cross-sectional area (Häggmark et al. 1978); probably also mirroring the volume of the examined muscle. Our data can therefore be interpreted to imply that fiber type composition and selective FT fiber hypertrophy are both important factors for muscle force production at slow velocities as well as during high speed contractions (Tihanyi et al. 1982) as suggested by Coyle et al. (1979) Gregor et al. (1979): This permits a refinement of the general view that muscle mass per se is the predominant factor influencing force production (Ikai and Fukunaga 1972). This pattern was recently confirmed in a sample of more than 100 physically active subjects (Tesch, unpublished observations).

In summary, both fatigue and recovery of knee extensor muscles were found to correlate with the capillary density of that muscle. Since lactate release to the blood stream was

correlated with capillary supply, it is suggested that recovery processes are influenced by the rate of lactate disappearance from the muscle

Acknowledgements

The views and opinions contained in this report are those of the authors and are not to be construed as official or as reflecting the views of the Department of the US Army.

References

Andersen, P., Henriksson, J.: Capillary supply of the quadriceps femoris muscle of man: adaptive response to exercise. J. Physiol. 270, 677-690 (1977)

Asmussen, E.: Muscle fatigue. Med. Sci. Sports and Exercise. 11, 313-321 (1979)

Asmussen, E., Mazin, B.: Recuperation after muscular fatigue by "Diverting Activities". Eur. J. Appl. Physiol. 38, 1-7 (1978a)

Asmussen, E., Mazin, B.: A central nervous component in local muscular fatigue. Eur. J. Appl. Physiol. 38, 9-15 (1978b)

Baldwin, K.M., Tipton, C.M.: Work and metabolic patterns of fast and slow twitch skeletal muscle contracting in situ. Pflügers Arch. 334, 345-356 (1972)

Beatty, C.H., Peterson, R.D., Bocek, R.M.: Metabolism of red and white muscle fiber groups. Am. J. Physiol. 204, 939-942 (1963)

Bergström, J.: Muscle electrolytes in man. Scand. J. clin. Lab. Invest. (Suppl.) 68 (1962)

Brodal, P., Ingjer, F., Hermansen, L.: Capillary supply of skeletal muscle fibers in untrained and endurance-trained men. Am. J. Physiol. 232, H705-H712 (1977).

Coyle, E.F., Costill, D.L., Lesmes, G.R.: Leg extension power and muscle fiber composition. Med. Sci. Sports. 11, 12-15 (1979)

Edwards, R.H.T.: Human muscle fatigue: physiological mechanisms. Pitman Medical, London (Ciba Foundation Symposium 82) p. 1-18 (1981).

Folkow, B., Halicka, H.D.: A comparison between "red" and "white" muscle with respect to blood supply, capillary surface area and oxygen uptake during rest and exercise. Microvascular Research. 1, 1-14 (1968)

Gregor, R.J., Edgerton, V.R., Perrine, J.J., Campion, D.S., Debus, C.: Torque-velocity relationships and muscle fiber composition in elite female athletes. J. Appl. Physiol. 47, 388-392 (1979)

Häggmark, T., Jansson, E., Svane, B.: Cross-sectional area of the thigh muscle in man measured by computed tomography. Scand. J. clin. Lab. Invest. 38, 355-360 (1978)

Hermansen, L.: Effect of metabolic changes on force generation in skeletal muscle during maximal exercise. Human muscle fatigue: physiological mechanisms. Pitman Medical, London (Ciba Foundation Symposium 82) p. 75-88 (1981).

Hudlická, O.: Growth of capillaries in skeletal and cardiac muscle. Circulat. Res. 50, 451-461 (1982)

Ikai, M., Fukunaga, T.: Calculation of muscle strength per unit cross-sectional area of human muscle by means of ultrasonic measurement. Int. Z. angew. Physiol. Einschl. Arbeitsphysiol. 26, 26-32 (1968)

Ivy, J.L., Sherman, W.M., Miller, J.M., Maxwell, B.D., Costill, D.L.: Relationship between muscle $\dot{Q}O_2$ and fatigue during repeated isokinetic contractions. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 53, 470-474 (1982)

Jorfeldt, L., Juhlin-Dannfelt, A., Karlsson, J.: Lactate release in relation to tissue lactate in human skeletal muscle during exercise. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 44, 350-352 (1978)

Karlsson, J.: Localized muscular fatigue: Role of muscle metabolism and substrate depletion. In: Exercise and Sport Sciences Reviews, Vol. 7. The Franklin Institute Press 1979

Kugelberg, E., Edström, L.: Differential histochemical effects of muscle contractions on phosphorylase and glycogen in various types of fibres: relation to fatigue. J. Neurol. Neurosurg. Psychiat. 31, 415-423 (1968)

Kugelberg, E., Lindegren, B.: Transmission and contraction fatigue of rat motor units in relation to succinate dehydrogenase activity of motor unit fibres. J. Physiol. 288, 285-300 (1979)

Nakamura, Y., Schwartz, A.: The influence of hydrogen ion concentration on calcium binding and release by skeletal muscle sarcoplasmic reticulum. J. Gen. Physiol. 59, 22-32 (1972)

Nassar-Gentina, V., Passonneau, J.V., Vergara, J.L., Rapoport, S.I.: Metabolic correlates of fatigue and of recovery from fatigue in single frog muscle fibers. J. Gen. Physiol. 72, 593-606 (1978)

Novikoff, A.B., Shin, W.Y., Drucker, J.: Mitochondrial localization of oxidative enzymes: staining results with two tetrazolium salts. J. Biophys. Biochem. Cytol. 9, 47-61 (1961)

Padykula, H.A., Herman, E.: The specificity of the histochemical method for adenosine triphosphatase. J. Histochem. Cytochem. 3, 170-195 (1955)

Robertson, S.P., Kerrick, W.G.L.: The effects of pH on Ca^{2+}-activated force in frog skeletal muscle fibers. Pflügers Arch. 380, 41-45 (1979)

Sigma Technical Bulletin 826. St Louis, Mo.: Sigma Chemical Co. 1968

Simonson, E.: Physiology of work capacity and fatigue. Springfield, Illinois: Charles C. Thomas 1971

Sjödin, B.: Lactate dehydrogenase in human skeletal muscle. Acta Physiol. Scand. (Suppl.) 436 (1976)

Tesch, P.: Muscle fatigue in man. Acta Physiol. Scand. (Suppl.) 480, 1-90 (1980)

Tesch, P., Sjödin, B., Karlsson, J.: Relationship between lactate accumulation, LDH activity, LDH isozyme and fibre type distribution in human skeletal muscle. Acta Physiol. Scand. 103, 40-46 (1978)

Tesch, P.A., Wright, J.E., Daniels, W.L., Sjödin, B.: Physical performances and muscle metabolic characteristics. In: Proc. Fifth International Symposium on the Biochemistry of Exercise, Boston (1982)

Thorstensson, A., Karlsson, J.: Fatiguability and fibre composition of human skeletal muscle. Acta Physiol. Scand. 98, 318-322 (1976)

Tihanyi, J., Apor, P., Fekete, Gy.: Force-velocity-power characteristics and fiber composition in human knee extensor muscles. Eur. J. Appl. Physiol. 48, 331-343 (1982)

TABLE 1. Mean (±SD and range) values for physiological and histochemical variables examined.

Physiological variables	Mean (±SD)	Range
Peak torque at $30^{o} \cdot s^{-1}$, $Nm \cdot kg^{-1}$ b.w	3.7 (±0.8)	1.7-5.1
-"- $180^{o} \cdot s^{-1}$, -"- -"-	2.3 (±0.4)	1.3-3.1
-"- $300^{o} \cdot s^{-1}$, -"- -"-	1.6 (±0.3)	1.3-2.1
Fatigue, % peak torque decrease	67 (±11)	47-82
Recovery, % peak torque of initial	70 (±13)	47-86
Histochemical variables		
Fiber type distribution, % FT	46 (±12)	19-60
-"- -"- , % FT area	51 (±15)	18-66
Mean fiber area, $\mu m^{2} \cdot 100$	66 (±18)	39-108
Capillary density, $cap \cdot mm^{-2}$	274 (±85)	156-489
-"- -"- , $cap \cdot fib^{-1}$	1.65(±0.30)	1.12-2.24

TABLE 2. Relationships between knee extension peak torque at different angular velocities, expressed in absolute and relative (to body weight) terms, and mean fiber area and fiber type distribution. Level of significance is denoted ($p<0.05$=*, $p<0.01$=**, $p<0.001$=***, $p>0.05$=n.s).

		Mean fiber area	% FT area
Peak torque	$30^{o} \cdot s^{-1}$, Nm	0.47 n.s	0.48 n.s
-"-	-"- , $Nm \cdot kg^{-1}$	0.46 n.s	0.57 *
-"-	$180^{o} \cdot s^{-1}$, Nm	0.54 *	0.61 **
-"-	-"- , $Nm \cdot kg^{-1}$	0.55 *	0.82 ***
-"-	$300^{o} \cdot s^{-1}$, Nm	0.09 n.s	0.57 *
-"-	-"- , $Nm \cdot kg^{-1}$	0.41 n.s	0.77 ***

Figure legends

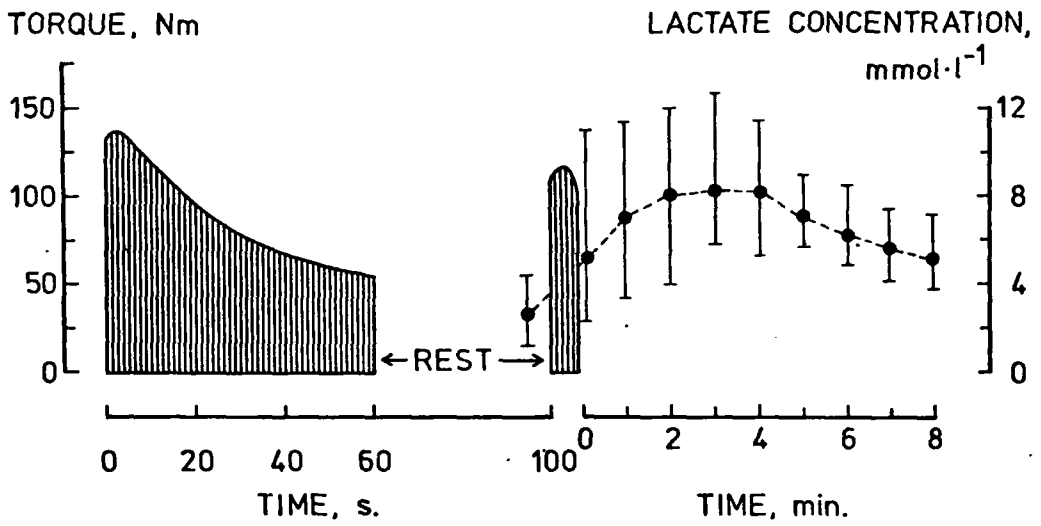
Fig. 1. Schematic description of the experimental design showing force production during exercise and following a 40 s rest period. Values for lactate concentration are mean and range.

Fig. 2. Examples of post exercise lactate response in two subjects. Lactate release index was calculated as the lactate concentration immediately post the fifth contraction relative to peak lactate concentration during recovery. From the picture it is evident that lactate release is different in the two individuals exemplified (i.e. a greater lactate release is indicated by filled dots as compared with open dots).

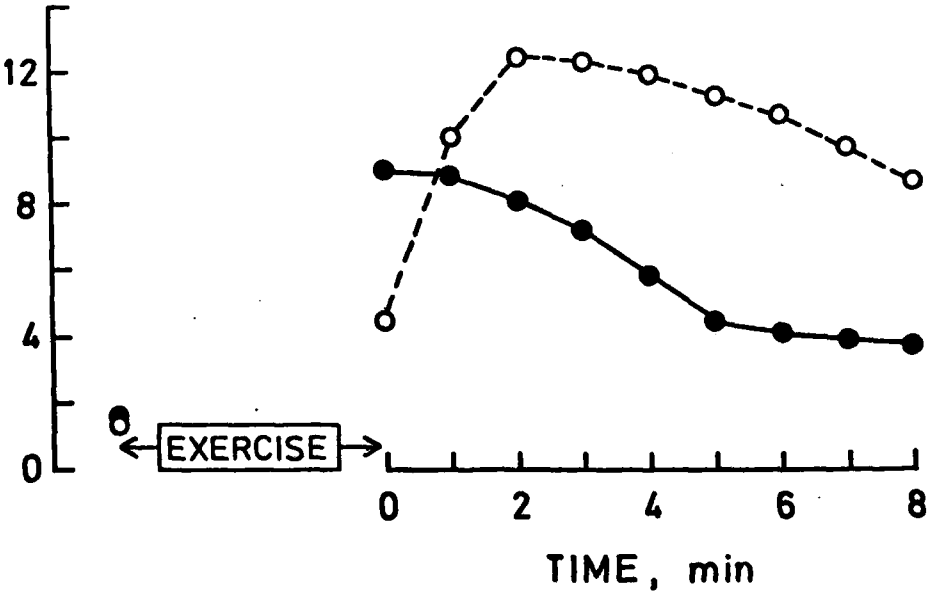
Fig. 3. The relationship of recovery to capillary density.

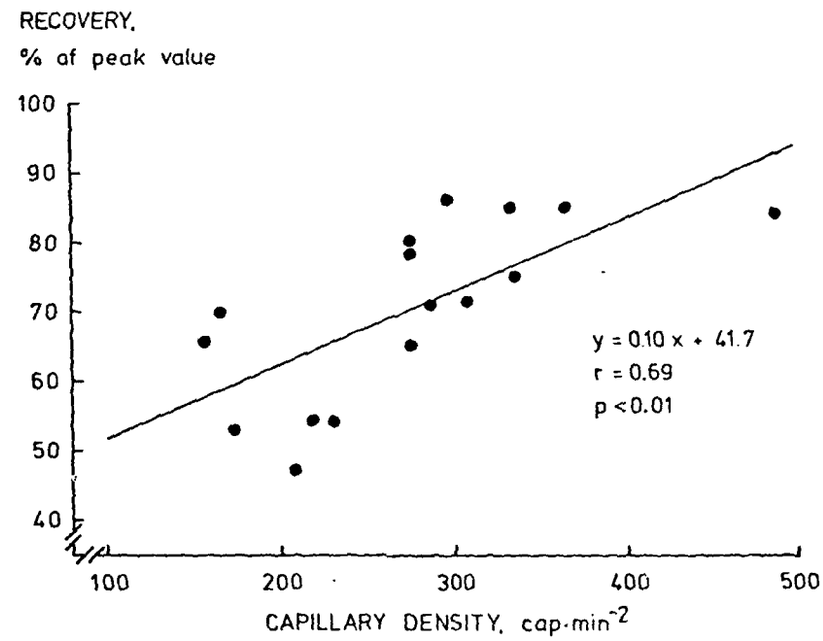
Fig. 4. The relationship of lactate release to capillary density.

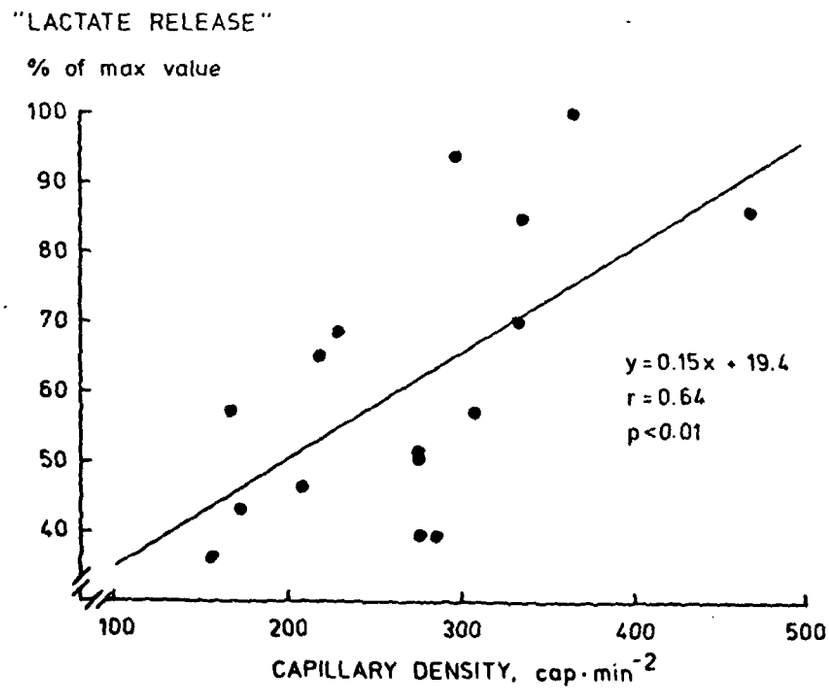
EXPERIMENTAL DESIGN.



BLOOD LACTATE CONCENTRATION,
mmol·l^{-1}




RECOVERY,
% of peak value
100
90
80
70
60
50
40
100
200
300
400
500
y = 0.10 x + 41.7
r = 0.69
p < 0.01
CAPILLARY DENSITY, cap·min-2


"LACTATE RELEASE"
% of max value
100
90
80
70
60
50
40
100
200
300
400
500
y = 0.15x + 19.4
r = 0.64
p<0.01
CAPILLARY DENSITY, cap·min-2

HUMAN RESEARCH

Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

The views, opinions, and/or findings contained in this report are those of the author(s) and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation.

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